sweating and the increased nitroglycerin requirement, a blood specimen was taken for determination of protein-bound iodine and uptake of radioactive triiodothyronine and the patient was admitted to hospital. Intensive efforts to relieve the nausea with symptomatic treatment were unsuccessful. An electrocardiogram in the office before admission did not show changes characteristic of digitalis intoxication. An x-ray film of the chest in the hospital was normal except for calcific plaques in the aortic arch. An upper gastrointestinal tract series was normal except for slight irregularity in the gastric antrum and some indentation of the post-bulbar duodenum. No abnormalities were observed in barium enema studies or in sigmoidoscopic examination. Results of a complete blood count and urinalysis were within normal range. Serum cholesterol was 107 mg, fasting glucose 108 mg and urea nitrogen 22.5 mg per 100 ml. Thymol turbidity was 4.0 units, bromsulfalein retention less than 2 per cent, lactic dehydrogenase 365 units, sodium 148 mEq and potassium 5.6 mEq per liter, zinc turbidity 6 units, and prothrombin time 17 seconds with a control of 16 seconds. Shortly after the patient was admitted, a telephone report was received that the protein-bound iodine content of the specimen of blood taken just before admittance to hospital was 14 micrograms per 100 ml and the uptake of radioactive triidothyronine was 36 per cent. No additional abnormalities were elicited in the course of examination. On January 16, radioactive iodine uptake in 24 hours was 88.7 per cent, and on January 23 the serum protein-bound iodine was 13.7 micrograms per 100 ml.

The patient was referred to a radiotherapist who recommended administration of propylthiouracil for three weeks, then a therapeutic dose of I^{131}.* This was begun, and when the patient was seen on February 15, after three weeks of treatment with propylthiouracil, angina had decreased, his appetite had improved, and bowel movements had decreased in frequency. He said that he had noticed a decrease in nausea within 24 hours after starting to take propylthiouracil, and complete disappearance of this symptom and of angina after another 48 hours. Then just before administration of the therapeutic dose of I^{131}, he was advised to discontinue propylthiouracil temporarily. Angina recurred almost immediately and within three or four days he again had chronic, moderately severe intractable nausea. Propylthiouracil was resumed but it had to be discontinued when arthralgia developed, and once more nausea recurred and angina increased. Administration of methimazole (Tapazole®) relieved the nausea and angina, and after the therapeutic dose of I^{131} was given the patient recovered and remained symptom-free.

Sheldon Margen, M.D., carried out the radioactive tracer studies and the therapeutic administration of iodine 131.

Discussion

Although interesting because of the rather occult nature of hyperthyroidism in this patient and the presence of multiple other problems, the main point of interest in this case must be the striking correlation of chronic nausea with hyperthyroidism, the relief of this symptom when thyrotoxicosis came under control and recurrence of the symptom whenever treatment was interrupted. This is a rather rare manifestation of hyperthyroidism, but an important one to remember.

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Exogenous Adrenal Suppression in Infancy

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THE ABILITY of the normal adrenal gland to survive long periods of suppression has been investigated previously.[1] Not to my knowledge, however, have the consequences of suppression of the neonatal adrenal gland for prolonged periods been analyzed. An amazing set of circumstances gave me an opportunity to study adrenal gland suppression in an infant and the results contribute to the known facts concerning the ability of the adrenal-pituitary axis to withstand prolonged suppression.

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TABLE 1.—*Results of Laboratory Studies of Plasma and Urinary Contents Associated with Intermittent Hormonal Therapy*

Date	Hormonal Therapy: Cortisone (daily)	Hormonal Therapy: ACTH	Urine: Total Volume (ml)	Urine: Creatinine (mg/100 ml)	Urine: 17-hydroxycorticoids* (mg/24 hours)	Urine: 17-ketosteroids (mg/24 hours)	Urine: Pregnanetriol (mg/24 hours)	Plasma 17 OHCS (μgm/100 ml)
December 1963—								
9	10 mg		470	148	5.4	1.1	0.04	.....
15	10 mg	5 U q 12 hours × 5 days	960	.....	5.3	1.1	0.4	.....
January 1964—								
5	10 mg	15 U q 2 days × 30 days	450	171	4.6	1.0	0.4	.....
8	Dexamethasone 0.33 mg (1 day)	15 U q 2 days × 33 days	405	191	5.1	1.0	1.0	.....
31	0 × 2 days	0 × 3 weeks	570	108	2.1	0.0	....	.....
February—								
2	0 × 7 days	0 × 3 weeks	830	.....	2.9	0.2	0.1	.....
1	8 a.m.		.....	.....	....	....	....	17.2
1	4 p.m.		.....	.....	....	....	....	7.0

*Porter-Silber chromagens in the urine.

Report of a Case

The patient, a 21-month-old white boy, was the fourth of six children. Four siblings were living and well. The fifth died at eight weeks following severe gastroenteritis (a diagnosis of salt-losing adrenogenital syndrome was made on the basis of biochemical evidence and confirmed at autopsy). A maternal cousin, aged 12, also had congenital virilizing adrenal hyperplasia.

He was born in India, far removed from modern laboratory facilities. When he was ten days of age, vomiting, weight loss, weakness and lethargy developed. The local physician, aware of the family history, made a presumptive diagnosis of salt-losing adrenal hyperplasia and treated the infant with cortisone, desoxycorticosterone acetate and the addition of salt to the diet. No stigmata of virilization were noted at the onset of therapy. The child thrived on this regimen, with normal rate of growth, weight increment and mental achievement. At seven months of age, periorbital and distal extremity edema was noted and salt addition was discontinued. Minor infections did not cause undue difficulty and the desoxycorticosterone acetate was reduced and finally discontinued when the patient was 16 months of age. Cortisone therapy was increased gradually by adding approximately 5 mg to the daily dose every three months until, at the time I first saw him, he was receiving 37.5 mg daily in divided doses. No signs or symptoms of Addisonian crisis or Cushing's syndrome have ever been observed. The patient was admitted to H. C. Moffitt Hospital at 21 months for evaluation of adrenal status.

On physical examination he appeared well developed, well nourished, alert, active and intelligent, and there was no evidence of acute or chronic disease. His height (85.5 cm) and weight (29 kg) approximated the 50th percentile for his chronological age. Blood pressure was 115/75 mm of mercury, the pulse rate 100 and respirations 22 per minute. Specifically noted was the absence of moon face, centripetal fat distribution, buffalo hump, striae, acne, hirsutism, sexual hair, phallic enlargement, abnormal rugation of the scrotum, wasting and neurological abnormality.

The results of laboratory studies of serum and urinary contents are shown in Tables 1 and 2.

Results of routine examination of the blood were as follows: Hemoglobin, 13.2 gm per 100 ml, hematocrit 40 per cent; leukocytes 7,000 per cu mm—40 per cent polymorphonuclear cells, 4 per cent eosinophils, lymphocytes 52, monocytes 2. The specific gravity of the urine was 1.015, the pH 5.5. There was no evidence of sugar, acetone or albumin, and the sediment was within normal limits.

Roentgenograms of the skeleton showed a bone age of 18 months at a chronological age of 21 months. An intravenous pyelogram and renotomograms showed no abnormalities and retrograde renal air insufflation showed no evidence of adrenal tumor.

During a three-month period of observation, reduction of cortisone from 37.5 mg daily to 10 mg daily had no noticeable clinical or biochemical effect. Stimulation with ACTH three times weekly for one month was similarly accompanied by no change. Cessation of all medication did not cause any adverse signs or symptoms during a

TABLE 2.—*Laboratory Data on Serum and Urinary Contents Associated with Hormonal Therapy*

Date 1964	Treatment	K	Na	Cl	CO_2	Creatinine	Ca	Total Protein
		mEq/Liter				(mg/100 ml)		(gm/100 ml)
SERUM:								
January—								
8	10 mg cortisone + 15 U ACTH daily	5.0	137	.....	.....	....	.....	....
30	0 × 2 days	5.0	141	114	.....	0.5	.....	....
February—								
4	0 × 7 days	5.2	135	106.5	21.8	0.6	10.7	7.2
URINE:								
January—								
7	10 mg cortisone + 15 U ACTH (450 ml)	33	36	.....	.....	....	.....	....
10	10 mg cortisone + 15 U ACTH (405 ml) sp. grav. 1.019	24.3	49.4	62.0	.....	68	.....	....

ten-day period and the moderate stress of the retroperitoneal air insufflation study was well tolerated.

Comment

The degree of suppression achieved during the 21 months of life in the patient in the present case is unknown, but the absence of any features of Cushing's syndrome or growth failure suggests that suppression might not have been complete. Nevertheless, the dosage of cortisone was always greater than the 15 to 25 mg per square meter of body surface which is assumed to be the average adrenal secretory rate. It thus seems reasonable that a considerable degree of suppression was present at all times.

The relatively high values of 17-hydroxycorticosteroids (Table 1) probably represent the combined total of the degradation products of endogenously secreted cortisol and the exogenous 10 mg of cortisone that the patient was then receiving. (Approximately 30 per cent of exogenously administered cortisone is excreted in the urine as Porter-Silber chromogens.)

The one elevated level of urinary 17-hydroxycorticosteroids while the patient was receiving dexamethasone is difficult to understand. Cessation of therapy demonstrated the low normal level of urinary 17-hydroxycorticosteroids produced endogenously. Unfortunately, an ACTH stimulation test was not attempted at that time. However, the demonstrable diurnal variation of plasma cortisol levels within the normal range strongly suggests the preservation of pituitary ACTH secretion and the ability of the adrenal glands to respond. The capability of this system to respond to severe stress was not tested.

There is no evidence to support the diagnosis of adrenal adenomata or adrenal virilizing hyperplasia in this patient. Steroid supplementation was advised for any acute stress during the year following the time the patient was under observation.

Continuous suppression of the adrenal gland of a boy baby for almost two years beginning soon after birth did not result in irreversible disuse-atrophy of the gland or in permanent loss of pituitary ACTH secretion.

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Persistent Mediastinal Hematoma

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INTRAPULMONARY HEMATOMA subsequent to thoracic trauma is a well recognized and documented entity. Localized and circumscribed collections of blood are often seen roentgenographically following closed-chest injury. In some instances such hematomas have been mistaken for intrapulmonary neoplasms or granulomas. As a consequence of the usual shrinkage and eventual

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